

# Should patients with active foot ulcers be non-weight bearing or take exercise to improve cardiovascular fitness?



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In focusing on foot ulcer healing and amputation prevention, are clinicians failing to address the more common, potentially modifiable, adverse outcomes of vascular associated early death and morbidity which can be affected by exercise? Ketan Dhatariya argues that people with active ulcers should be non-weight bearing, while Martin Fox argues the case for exercise.

## The case for non-weight bearing

It has been well recognised for many years that the presence of a diabetic foot ulcer is associated with premature mortality (Brownrigg et al, 2012). In 2003, Moulik et al reported that the presence of a foot ulcer was associated with a worse 5-year survival than either breast or prostate cancer. However, unlike those malignancies, the predominant cause of death in individuals with diabetic foot ulcers is macrovascular disease in the form of cerebrovascular disease and myocardial infarction. The foot ulcer is merely the culmination and external manifestation of several underlying conditions – most of which are likely to have been present for many years.

The triad of risk factors that pre-dispose somebody to developing a foot ulcer are neuropathy, ischaemia and pressure. The contribution that each of these factors makes to developing a foot ulcer has been well described in the literature (Jeffcoate and Harding, 2003). Other factors include advancing age and trauma. Of course, neuropathy may lead to foot deformity, which increases the direct pressure and shear stresses on the vulnerable parts of the foot (Cavanagh et al, 2008). Because of this, offloading remains a cornerstone of treatment, but questions remain as to how long to remain non-weight bearing, the best way to achieve pressure relief, how much pressure on the foot is too much, and so on. However, for uncomplicated wounds, offloading has been shown to be beneficial (Cavanagh and Bus, 2011). Finally, there is the separate issue of

superadded infection, which is usually a secondary occurrence once the skin has been breached (Jeffcoate and Harding, 2003). It is the lack of blood supply, however, that is often the underlying major risk factor for ulceration and amputation (Boulton et al, 1999; Hinchliffe et al, 2008).

The principles of treatment of diabetic foot ulcers have remained unchanged for many years. Debridement of the wound to get rid of any non-healthy tissue which may be impeding healing; ensuring that the foot has adequate blood supply with re-vascularisation as necessary; pressure relief either in combination with debriding and removal of callus but also using offloading orthoses, removable below-knee walking boots, or a total contact plaster cast; and antibiotics for infection (NICE, 2011). This combined approach has been used to treat the most pressing issues associated with the ulcer – the worst case scenario being a limb-threatening wound.

The origins of macrovascular disease in diabetes are complex, but involve the interaction between dysfunctional endothelial cells, hypertrophied vascular smooth muscle cells, overactive platelets, and other factors (Beckman, 2002). It is likely that the underlying atherosclerotic process has been ongoing and progressive for many years prior to the development of symptomatic peripheral vascular disease or an ulcer forming.

Previous work has shown that people with diabetes are two to four times more likely to develop peripheral arterial disease than those without diabetes and as a result are less likely to have pedal pulses (Abbott et al, 1990; Newman et al, 1993). In addition, the presence of neuropathy usually predates the onset of neuropathic or neuro-ischaemic ulcers. The neuropathy changes the muscle tone within the foot, resulting in changes in biomechanics and subsequent abnormal pressure distributions (Bus et al, 2002; Cavanagh et al, 2008). By the time the ulcer appears, these contributing factors are usually well-established. Thus, it would be more appropriate

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to say that – as with most things in the world of diabetes – prevention of these complications would be much better than cure. Therefore to increase activity levels to try and improve cardiovascular fitness after the development of foot ulcers is rather like closing the stable door after the horse has bolted.

In the USA, the mean age of diagnosis of diabetes over the last 10 years has been steady at about 54 years old (Centers for Disease Control, 2014). This includes those with type 1 diabetes, thus it is likely that the vast majority of people with type 2 are diagnosed in their 60s. The same data set shows that over 40% of this age group with diabetes are physically inactive (Centers for Disease Control, 2014). I would venture to suggest that physical inactivity in their youth contributed in part to the development of diabetes, and that once they had developed established complications they were even less likely to become more physically active.

It is time to hold my hand up and admit my failings – I am sure that however hard I try, I am unable to motivate the majority of my patients, even those newly diagnosed with type 2 diabetes, to eat less and exercise enough to delay the progression of their diabetes or prevent the onset of complications. Even if I do, the potential dangers of increased physical activity in people with established diabetic neuropathy have recently been highlighted by Gooday et al (2014) in an article describing individuals who had weight loss surgery and became physically more active as a result, but then went on to develop Charcot joints.

There has been data in the literature suggesting that multiple risk factor intervention in terms of optimising blood pressure, lipids and glycaemic control, helps to prevent lower extremity ulceration, gangrene or amputation and also reduces mortality (Malik et al, 2013; Young et al, 2008). It may well be that the introduction of adjunctive therapies over the last 10 years or so has been responsible for the reported general decline in amputation rates (Krishnan et al, 2008). However, there is not universal agreement that this approach works (Morbach et al, 2012). Despite this apparent lack of convincing evidence for risk factor optimisation, brave is the clinician who does not address these issues in the foot clinic! What is acknowledged is that these interventions address ulcer prevention, but that once an individual has developed an active

ulcer the issue of “risk” is no longer appropriate – they already have developed the condition, and therefore management should focus on the treatment of the ulcer. Of course, at the same time as the ulcer is being addressed, the long-term risk factors should also be managed because people usually die of premature cardiovascular disease. In addition to optimisation of glucose, blood pressure and lipids, these interventions could include exercise to improve cardiovascular fitness.

The evidence for offloading ulcers is strong. There have been several trials to show that using non-removable casts or non-removable below-knee walking boots increases the proportion of ulcers that heal, and also speeds up healing times compared to removable devices or shop-bought shoes (Mueller et al, 1989; Margolis et al, 1999; Caravaggi et al, 2000; Armstrong et al, 2001; Armstrong et al, 2005; Piaggese et al, 2007). There is poor compliance with removable devices – with one famous study showing that removable devices were only worn for 28% of the time that the patient was active (Armstrong et al, 2003). If patients do not wear their removable prescribed footwear, then their healing time is slower compared to those who are continuously offloaded. However, despite this evidence, it is clear that most specialist foot clinics do not offer non-removable offloading devices (Prompers et al, 2008; Wu et al, 2008).

The suggestion that in addition to offloading, exercise should be included as part of the “multiple risk factor intervention” strategy raises several questions. In individuals with diabetes and existing ulcers, does exercise help? And if so, what are we trying to prevent? If the goal is prevention of premature cardiovascular death, does the addition of an exercise regimen have significant benefits beyond traditional risk factor management? Does exercise help wounds heal faster? Or does the elevated pressure due to increased weight bearing with exercise delay healing? How much exercise does one have to do to see a health benefit? What sort of exercise would be of most benefit or suitable for people with active foot disease? How many people have to exercise and for how long to delay a single premature death (numbers needed to treat)? What are the risks of increasing exercise in terms of precipitating cardiovascular morbidity and mortality in a previously inactive person (numbers needed

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to harm)? There are data and recommendations for the general population (Wen, 2011), but how does that translate for any given individual with a diabetes-related foot ulcer? The data to answer these questions are not currently available. Does this mean that because it is an evidence free zone we should not encourage it?

In summary, the data to support the notation that exercise is beneficial for people with diabetes-related foot disease is lacking, and there remains the fear of either worsening active disease or even precipitating a cardiovascular event. As always, a lot of work remains to be done.

**The case for exercise**

Since the emergence of specialised diabetic foot clinics in the UK nearly 30 years ago (Edmonds et al, 1986), the collective mindset has been about preventing amputation and saving limbs. This has become the almost universal focus and goal of NHS multidisciplinary foot teams and foot protection teams – teams that have become the true champions and pioneers of best treatment of people with diabetes related foot disease. But here lies the problem. In focusing almost entirely on foot ulcer healing and amputation prevention, we have seemingly failed to accept and address the more common and potentially modifiable adverse outcomes of vascular associated early death and morbidity (Morbach et al, 2012). It gets worse! Our current “flat earth” approach to diabetic foot disease management may not only be failing to tackle the high vascular-related death rates in our patients, it may also be contributing to it. We are possibly denying people with diabetic foot disease and ulcers the very best of vascular risk management. In a nutshell – in trying to save more limbs, are we inadvertently killing our patients?

About 10 years ago, while helping to draft some regional podiatry guidelines on diabetic foot disease as the clinical lead of a high-risk foot team, I argued robustly with a very respected colleague that cardiovascular risk factor management was not my responsibility and was someone else’s job. My job was to save feet and legs. But around that time, NICE published its first guidelines on managing diabetic foot disease and cardiovascular risk management was clarified as a responsibility of the multidisciplinary foot team (NICE, 2004). A few years later, Young et al (2008) published results from their efforts to focus

on cardiovascular risk management medication with a cohort of their patients with diabetic foot ulcers, showing a dramatic improvement in 5-year mortality outcomes.

It took me until then to realise that I, the clinicians I was leading in my team and the majority of my peers and colleagues in the diabetic foot clinical community, were simply not aggressively tackling associated vascular risks. Certainly not with the same gusto that we were debriding, offloading, fighting infection and dressing the foot ulcers we were perhaps seduced and blinded by, on a daily basis. Our perspective was skewed and one-dimensional. Our earth was decidedly flat. We were disregarding the available, related evidence base.

Around 15% of people with diabetic foot ulcers may have lost a leg after 10 years, but up to 70% will have died and over half of these deaths are vascular related (Morbach et al, 2012). However, despite broad awareness by clinicians of these vascular related outcomes and 6 years on from Young et al’s 2008 article, I am still struggling to find diabetic foot teams who are promoting and providing key vascular interventions or publishing on the theme of vascular risk management. We are still very limb-focused, perhaps limb-obsessed, with our time and resources. Vascular risk management surely needs to be at the heart of all diabetic foot disease intervention, not an afterthought, a back-covering exercise, or something we would work on if we had more time.

Talking of exercise, there is a general consensus and a growing evidence base that moderate aerobic exercise plays a major role in the prevention and control of diabetes-related health complications (Colberg et al, 2010). Looking more broadly at the evidence base for people with vascular disease who are at risk of morbidity and mortality, cardiac rehabilitation programmes have been associated with up to a 56% improvement in survival among patients after myocardial infarction and a 28% reduction in risk of recurrent myocardial infarction (Witt et al, 2004).

Recently in the UK and a high profile campaign has been launched around the theme of the “foot attack”, likening diabetic foot ulcers to heart attacks (Vig et al, 2014). It is perhaps ironic that the key intervention of cardiovascular exercise for people who have had heart attacks is the very same intervention that is actively withdrawn or at

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least discouraged in people who have a foot attack. People with diabetic foot ulcers or Charcot foot are universally advised by diabetic foot teams and others to rest the foot, thus ceasing any exercise or health and well-being activity, for weeks, months or often years. To actively deny people who have high risks of vascular related death from an intervention shown to reduce these risks, we must have a very robust counter evidence base and sound clinical reasoning, mustn't we? Where is this evidence base? Where does the unquestioned belief and culture of limiting physical activity come from?

There are two likely sources – the well-established evidence base for offloading plantar neuropathic foot ulcers and Charcot foot and also the general fear amongst diabetic foot teams that excess weight-bearing activity leads to foot ulceration, re-ulceration of healed feet, or deterioration of Charcot foot deformity.

While offloading plantar DFUs or Charcot foot in below-knee total contact casts is broadly accepted as beneficial (NICE, 2004), what is not clear is just how much offloading is required to heal an ulcer and how effective offloading is for anything other than plantar neuropathic ulcers. In Charcot foot, where total non-weight bearing has been promoted for many years in the initial acute phase, there is now some limited published evidence to suggest that a degree of protected weight bearing may be a safe treatment option after all (Parisi et al, 2013). Bearing in mind that the vascular-related mortality outcomes for people with Charcot foot are similar to those with foot ulcers (van Baal et al, 2010), is the concept of providing safe cardiovascular exercise for people with Charcot foot something we should also urgently consider? Does the need to provide offloading or immobilisation of foot and ankle joints exclude the possibility of also providing safe, effective vascular protective exercise? Or could we provide and promote both? Seated, upper body cardiovascular exercise programmes may be both safe and effective for people with active foot disease.

Despite the culture of fear that prevails around promoting activity or exercise for people with diabetic foot disease, the limited evidence base seems to contradict the current mantra of “rest to avoid harm”. It has been shown that exercise does not increase the incidence of diabetic foot ulcers in people with peripheral neuropathy (LeMaster et

al, 2008), and it does not increase re-ulceration in people who have healed from foot ulcers (LeMaster et al, 2003). There appears to be no significant published evidence demonstrating that supervised cardiovascular exercise is adversely linked with foot ulcers, Charcot foot or associated amputation. A fear of excessive weight bearing and activity contributing to poor foot outcomes seems to have influenced a whole generation of clinicians to reject the available evidence to the contrary and perhaps lead us to neglect the need for further good quality research and audit in this area.

Search “diabetic foot ulcer” on PubMed and over 10000 published studies are available. When “cardiovascular exercise” is added to the search, the number of published studies drops to 22. There is a complete lack of significant direct evidence to support or reject exercise interventions for people with serious diabetic foot disease – the big question is why not?

There are some clinicians who might suggest that foot ulcers and Charcot foot occur late in diabetes-related vascular disease and it may be too late to engage people who have had a foot attack in cardiovascular exercise. But if “after the event” is too late, why would NICE recommend cardiovascular exercise as part of a rehabilitation programme to everyone who has had an acute myocardial infarction, regardless of age (NICE, 2013)? Concordance with exercise interventions is likely to be a challenge for people with foot disease, just as it is with people with cardiac disease or peripheral arterial disease. NICE has acknowledged this and in its guidance suggests strategies for the NHS to adopt, strategies that may work for diabetic foot disease patients too.

Cardiovascular rehabilitation programmes have been commissioned and set up throughout the UK. They offer a range of interventions which, when offered together, result in significant improvement in outcomes around cardiovascular events and related death in people at risk.

Although not widely known or promoted, people with diabetes and peripheral arterial disease are specifically identified as target groups for such programmes, alongside people with primary cardiac disease (British Association for Cardiovascular Prevention and Rehabilitation [BACPR], 2012).

BACPR has recently made a suggestion that offers the diabetic foot clinical community a possible



practical way forward. It states: “It is recognised that asymptomatic people, including those with diabetes, identified at high cardiovascular risk require the same professional lifestyle intervention, and appropriate risk factor and therapeutic management. Existing cardiac rehabilitation services are in a strong position to evolve to provide care to include a wider spectrum of patient groups” (BACPR, 2012).

Rather than trying to set up separate exercise and lifestyle intervention programmes for people with diabetic foot disease, why don't we look at negotiating access to existing, well-established, cardiovascular rehabilitation programmes in the NHS?

Is it time that we in the diabetic foot clinical community took a more “round earth” view of our diabetic foot disease patients and looked at negotiating routine access to cardiac rehabilitation programmes? Promoting and offering supervised, tailored, safe, cardiovascular exercise to all patients with foot ulcers and Charcot foot, along with other vascular risk interventions, may help us reduce the appalling mortality and morbidity rates we currently see in this population. The cumulative associated evidence to suggest patients will benefit far outweighs the irrational fear we harbour and propagate about exercise causing harm. It is time we focus our collective efforts on saving more lives as well as more limbs. If we don't lead on implementing effective vascular risk reducing interventions for our patients, who will? ■

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#### Acknowledgements

Ketan Dhatariya thanks Catherine Goody and Rachel Murchison for their valuable comments on this manuscript